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Editors :

T. DALLING, M.A., M.R.C.V.S.

J. McCUNN, M.R.C.V.S., L.R.C.P.

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EDITORIAL

NEW METHODS AND TECHNIQUES

We draw the attention of our readers to two articles in this number of the Veterinary Journal. The first concerns the studies of Dr. Duran-Jorda on local hæmopoiesis in the alimentary tracts of domestic animals. Following his studies on the human gastric mucosa, both normal and abnormal, certain tentative conclusions were made concerning the origin of eosinophils and red blood corpuscles. It was not, however, until domestic animals were the subject of careful histological examination that sufficient evidence was forthcoming to confirm the previous observations. The author has shown that in the abomasum of the cow and the sheep there occurs a process of hæmopoiesis. This finding is of much importance, and the fact that the clearest evidence was obtained in the cow and sheep proves once again the great value of "comparative" work in our search for truth. We have many examples of valuable results from such studies. The part to be played in general science investigations pertaining to medicine by a study of animals in general is very patent: we often arrive at a conclusion by experiments and work on animals of an entirely different species than that on which the original observations were made. It is also interesting to note that when the exact picture is not presented by examinations of tissues of animals of different species, an explanation for the differences can also be found. The findings of Dr. Duran-Jorda open the way for further research work on the subject, and it is hoped they will be found useful to those in a position to pursue such investigations.

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The other article is that by A. B. Paterson on the diagnosis of the condition in cows commonly referred to as "bovine hypoglycæmic ketosis." The author shows how by the use of samples of milk it is possible to arrive at a fairly definite conclusion concerning the degree of ketosis present in the animal. It is pointed out that the terms "acetonæmia" and "ketonæmia" refer simply to abnormally high levels of acetone or ketone bodies in the blood, and do not signify that a special disease is present. It is well known, however, that the excessive formation of ketone bodies in the body is found associated with other symptoms soon after calving, e.g., lack of appetite, wasting, fall in milk yield, etc.: therefore a rapid and convenient method of diagnosing their presence, especially under such conditions, is of marked value. A perusal of this work shows how various preliminary tests were necessary before conclusions could be made as to the value of the test, and how the experimental findings were checked up with those in field cases. The object of our research work must largely be to enable the application of methods and techniques in the prevention and control of ill-health and the diagnosis of disease conditions when they arise. While many such control measures and methods of diagnosis have their origin in laboratories, and while, especially with the latter, the laboratory is still the place where the work has to be done, we welcome any method which can be carried out on the spot and which will ensure a considerable measure of success. A typical example is the use of the agglutination test in diagnosing "carriers" of pullorum disease (B.W.D.). The original technique necessitated the sending to a laboratory of a sample of blood from each breeding fowl in the flock and the searching of the flock for positive reactors when the results of the testing were known. By the introduction of the "rapid" test, now largely practised in this country, it is possible to obtain results on the farm and the disposal of reacting fowls within a few minutes of the completion of the test. Concerning the Rothera test in milk samples, the author modestly says, "it deserves wider application than it has hitherto received": veterinary practitioners who have opportunities to try out this test in milk will do well to use it as an aid to diagnosis. It is of much interest that the author is able to record that the various drugs used in the treatment of cows affected with "post-parturient hypoglycæmic ketosis" ("post-parturient dyspepsia") do not interfere with this test of milk samples.

General Articles

LOCAL HÆMOPOIESIS AS SEEN IN THE ABOMASUM OF THE COW AND SHEEP

By FREDERIC DURAN-JORDA, M.D. (Barcelona),
Director of Pathology Department at Ancoats Hospital, Manchester, 4; Late Chief of the Blood Transfusion Service of the Spanish Republican Army.

It is well known that in the human being there is a long-established relation between the gastric and intestinal diseases and hæmopoiesis. This relation is not only a clinical one, as it has been found that in certain blood diseases there is an accompanying atrophy of the histological structure of the gastric mucosa, for example, in pernicious anæmia. It has also been found that some intestinal obstructions produce macrocytic anæmia, and the appearance of the blood in sprue gives the same picture.[1 2 3 4]

We kept these facts in mind whilst making our study of hæmopoiesis, and were fortunate in finding a sarcoma of the stomach where we could see hæmopoiesis in full development.[5 6]

We then commenced a thorough examination of the gastric mucosa of man and different animals.

From studies of both normal and pathological human gastric mucosa, we observed two things.

(1) In the sarcomatous stomach we could see how the red blood corpuscles were formed by the growing of very tiny granules to fully-developed red blood corpuscles. (Figs. 1-2.)

(2) There were a few cells in the normal gastric mucosa with the same appearance as the cells which have been described as Russell bodies. In a few cases these cells were even more numerous and evident in post-mortem stomachs. (Figs. 3-6.)

From our investigations in man of different angiomas, of the cellular contents of the sero and hæmorrhagic thyroid cysts, a sarcoma of the stomach and sections from bone marrow and spleen, we found that the red blood corpuscles develop from the eosinophils. Our next step, therefore, was to find the origin of the eosinophils.

We could not accept that these cells came from the bone marrow as there were not enough eosinophils present in that organ to explain the total secretion of red blood corpuscles. After an investigation of the gastric and intestinal mucosæ of various mammals, however, we found the presence of eosinophils to be very marked in these organs, and they could be counted in millions. Eosinophils were present everywhere in these mucosæ, diapedising outside the glands, amongst the glandular cells, underneath the glandular cells, and also in the interglandular spaces, and by making careful observations of the human gastric mucosa we found that we could easily link the affinity between the eosinophils and the oxyntic cells. It has also been established that the oxyntic cells show the same ashes as the eosinophils after micro-incineration.[7]

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That the hydrochloric acid is produced by the oxyntic cells is a theory which is rapidly losing ground.

(1) Because no one has been able to prove that the protoplasm of the oxyntic cells contains any acid.[8]

(2) Because in some atrophic stomachs where there are no oxyntic cells present there is still free hydrochloric acid.[9]

(3) Because the granules of the oxyntic cells, instead of disappearing during the process of digestion or immediately afterwards, show a larger quantity of granules than before digestion occurred.

(4) Because the physiologists believe that the hydrochloric acid is produced by the reaction between the sodium chloride contents in the lumen of the gland and the bicarbonates present in the blood of the gastric capillaries.

To try to confirm our previous findings in man, we began to study the stomachs of different mammals.

Pig

We started with the stomach of the pig, as the extract from this animal's stomach has been used in the treatment of some anæmias. (Figs. 7-13.)

Its general morphology was very similar to the human stomach. There were oxyntic cells present and eosinophils, but in the inter-glandular connective tissue it was very easy to see oxyntic cells in the process of secreting red blood corpuscles, the cells having the same appearance as the cells described as Russell bodies. This was a similar process to that we had found in the normal human stomach, but it could be seen more easily in the pig than in the human gastric mucosa. In general, however, we were disappointed with the gastric mucosa of the pig, because we could not find any fundamental difference from the histology of the human gastric mucosa, so we commenced an investigation of the abomasum of the cow and sheep.

Cow

In the abomasum mucosa of this animal we came across the objective solution we were seeking, to demonstrate without doubt the relation between the oxyntic cells and hæmopoiesis, and also the relation between the oxyntic cells and eosinophil formation. It could be seen clearly that these gastric glands contained a large number of cells with a granulated protoplasm which took the eosin stain. These cells could be divided into two groups.

(1) A group which develops its hæmopoiesis in situ, meaning that the granules vary from the eosinophil granules to the size of red blood corpuscles.

(2) Another group of cells which become eosinophils to develop the blood-forming granules in the actual accepted hæmopoietic organs, the bone marrow and spleen.

In any section made from the abomasum of the cow there was an impressive presence of these very large cells containing an uncountable number of granules which were developing "in situ." When a certain stage of granular development has been reached and the granules are the size of red blood corpuscles, it becomes

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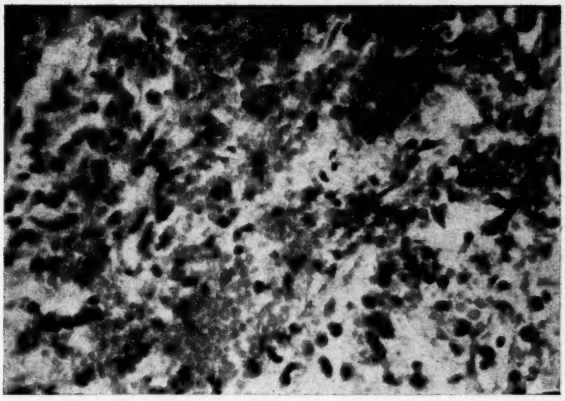
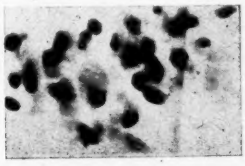
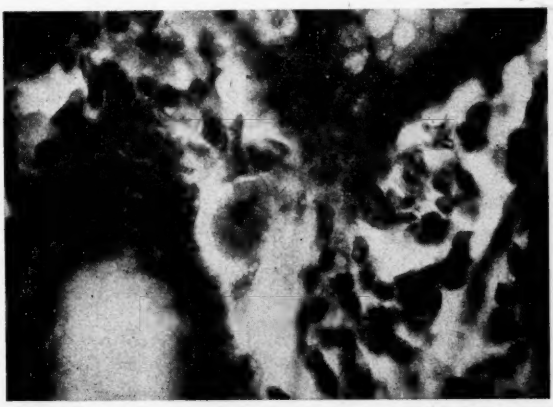


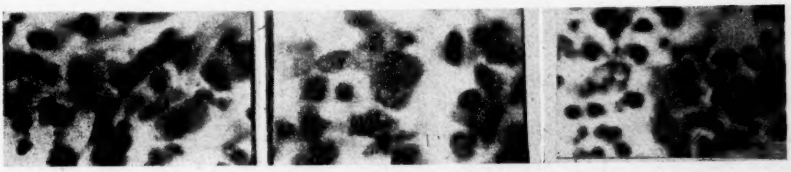
1. Sarcoma of stomach. Notice the different sizes of the granules.



2. Details of the sarcoma of stomach.



3. A blood-forming cell with the aspect of a Russell body cell.



4, 5, 6. Details of cells with the aspect of Russell bodies.

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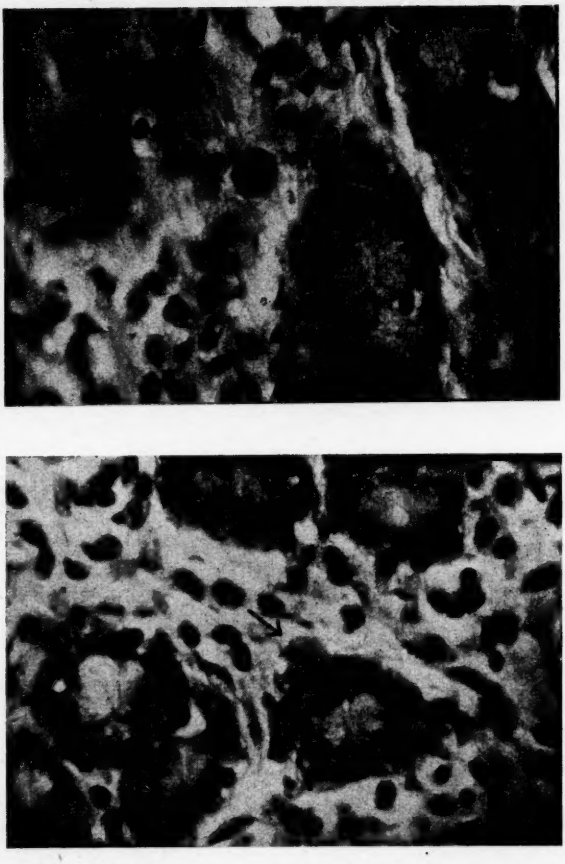
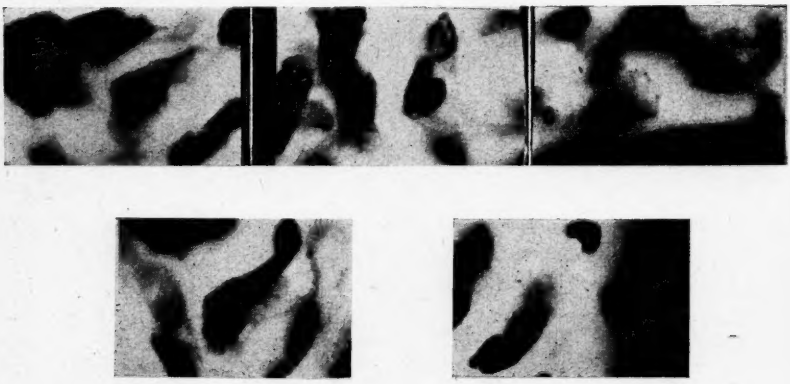
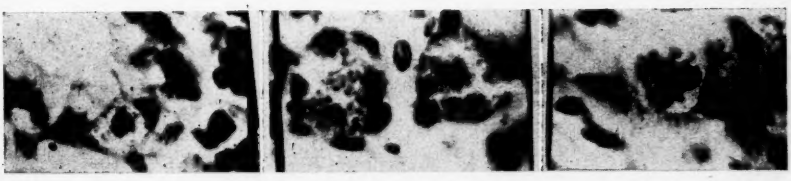


7, 8. Showing blood-forming cells in the inter-glandular connective space.

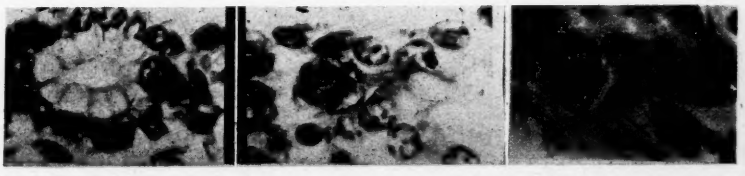


9-13. Details of different blood-forming cells.

14. Showing oxyntic cells and eosinophils in the abomasum.

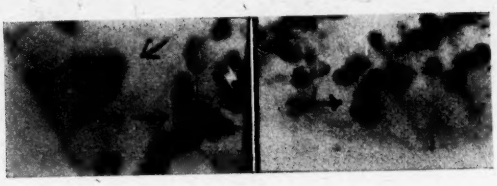


15-17. Details of the oxyntic cells secreting red blood corpuscles.



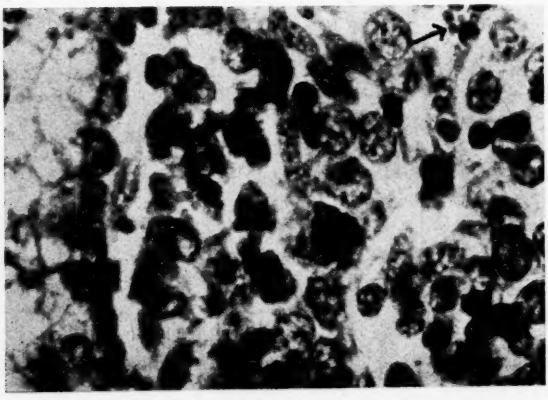
18-20. Showing the evolution of the oxyntic cell towards the eosinophil.

HUMAN FŒTUS.

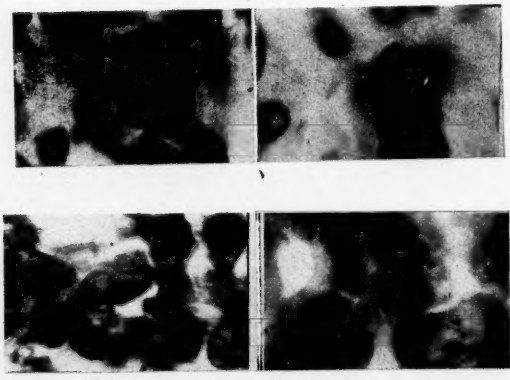


21-22. Basal kornel cells present in the human fœtus.

SHEEP.



23. Oxyntic cells secreting red blood corpuscles in the abomasum.



24-27. Oxyntic cells under high magnification.

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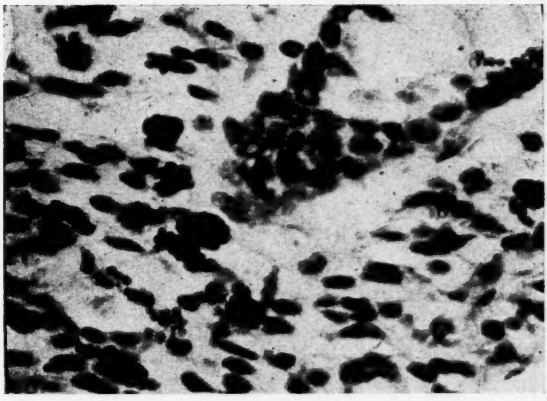
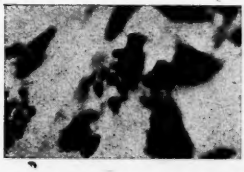


28. The large intestine of a horse showing blood-forming cells. This is very similar to the process found in sheep.



29, 30, 31, 32. Details of blood-secreting cells in the small intestine.

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difficult to distinguish whether it is a cell containing intra-protoplasmatic red blood corpuscles or a small capillary containing circulating red blood corpuscles. The only slight difference is that when stained with eosin the colour of the red corpuscles is more red if they are contained in the protoplasm of the cell than those circulating in a capillary, which are rather orange in colour, probably on account of the oxygenation of their hæmoglobin. (Figs. 14-17.)

It appears, however, that the granules do not develop altogether, but in small batches, which develop fully and are then expelled from the cell. This was in contrast to the process we had seen in the human thyroid cysts, where, floating in the cystic fluid, can be found very large cells developing all their intraprotoplasmatic granules into red blood corpuscles simultaneously, the cell having a monstrous size. The reason for this is probably a physical one. In the thyroid cysts the cells are swimming free and their growth is not inhibited in any way, but when the cells are established in a tissue their growth becomes limited by the neighbouring cells.[10]

It can also be studied in the abomasum mucosa that there is at the same time a process of evolution from the oxyntic cells to the eosinophils. (Figs. 18-20.) Some of the granules of the oxyntic cells become more agglutinated and small in size, and the cell, with the intermediary nuclear aspect between the oxyntic cell and the eosinophil polymorph, diapedises from the gland towards the sub-mucosa connective tissue. It is remarkable that the eosinophil cells have their nuclei nearer to the lumen of the gland than their granules, similar in aspect to the basal kornel cells found in the intestine of the human fœtus and in some small animals, especially the rat. (Figs. 21-22.)

Sheep

In the abomasum of the sheep can be found a very similar process to the one described in the same organ in the cow, but the cells appear to contain less granules, and consequently this allows the intra-cellular granules to grow simultaneously to the full size of red blood corpuscles. As I have said before, in some cases it is very difficult to distinguish them from those contained in a capillary, and they can only be identified by the difference in colour, as described previously. (Figs. 23-27.)

Horse

In the intestine of the horse we have confirmed a similar process to that found in the abomasum of the cow. (Figs. 28-32.)

Small Mammals

Studying the gastric mucosa of small animals, such as the rabbit, guinea pig and rat, these hæmopoietic processes, as seen in the cow and sheep, could not be found in the small mammals, and one can only find occasional cells secreting red blood corpuscles in situ in the same way as seen in man and pig.

As we have described fully elsewhere,[5 6] to complete the evolutive cellular cycle, the cell becomes a lymphocyte after it has secreted and expelled the full-size red blood corpuscles. The lymphocyte is again fixed in the gastric and intestinal mucosæ, when, in order to begin a new evolutive cycle, it recommences the preparation of granules in its protoplasm as an oxyntic cell.

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We are confident that a study of this hæmopoiesis as seen in the abomasum of the cow and sheep will give hæmatologists a clear understanding of the secretion of red blood corpuscles locally by the oxyntic cells, and in these gastric organs it can be demonstrated without any doubt that the eosinophil polymorph is, after all, a wandering, dehydrated oxyntic cell.

Having shown by our investigations the intimate connection which exists between the gastric cellular morphology and hæmopoiesis, we are hoping that the primary advantage of these investigations will be to take up the scientific study of diets to speed up the regeneration of the red blood corpuscles. Unfortunately, we have not the facilities to do this ourselves, as it should involve a close study of the two herbivora, the cow and sheep, and consequently we leave this research to those who are better able to do it than we are. We can also conclude from our observations that the mucosa from the abomasum of these animals may have stronger hæmopoietic properties than the ones already used.

Summary

The author has studied different mammal stomachs and has confirmed the relation between the oxyntic cell and hæmopoiesis, and also the relation between the oxyntic cell and the eosinophil, as he has previously described in the human gastric mucosa.

In the stomach of the pig the local hæmopoiesis is only shown by occasional oxyntic cells secreting red blood corpuscles, the cells having the aspect of what have been described as Russell bodies.

In the abomasum of the cow and sheep the author has discovered a very important process of hæmopoiesis in situ, which process is recommended for the objective study of red blood corpuscle secretion from the oxyntic cell.

The stomachs of the small animals show only occasional red blood corpuscle secreting cells, similar to the process described in the pig.

The author has also confirmed the fixation of the lymphocyte in these mammal stomachs, which is converted into an oxyntic cell at the commencement of the evolutive cellular cycle.

My thanks are due to Professor Wood Jones for his great interest and encouragement whilst we have been working on this research. To Inspector Wilfred Taylor, C.R.S.I., for his invaluable assistance in the selection of animal specimens. I must also thank my technicians:—Mr. Butcher, for his great help with the histological sections, and Mr. Jeffrey B. Dean, B.Sc., who has done all the photomicrographs.

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THE RELATIONSHIP OF BOVINE TO HUMAN TUBERCULOSIS

By J. W. RAINEY, C.B.E., M.R.C.V.S.

As was to be expected, American reactions were hostile to the suggestion that the effect of the eradication of tuberculous cows in the United States on the mortality from human tuberculosis had been over-estimated.

Our colleagues in the United States, backed as they were by unstinted finance, and the opportune fact that a reduction in the bovine population for a while was politically desirable, achieved a great technical feat in the eradication of the tuberculous cow.

This achievement was greatly facilitated by the naturally low incidence of bovine tuberculosis in most States as compared, say, with Britain, but it was nevertheless a remarkable performance, and it is not surprising that any observations that might seem to belittle the value of the feat should be bitterly resented.

Still more hostile, not only in the United States but elsewhere, and even, strange as this may seem, in Queensland, was the reaction to the suggestion that human exposure to the bovine tuberculous germ might increase human resistance to the myco-bacterium tuberculosis (hominis).

This reaction was almost identical, but in reverse, to the now almost forgotten objections of anti-vaccinationists to the claim that cow pox would strengthen human resistance to small-pox. It seems odd that what was believed with almost religious fervour concerning the role of the cow in small-pox should now be contested with equally religious fervour as regards the role of the cow in tuberculosis.

Hostility, however, in such a matter, is not enough; such scientific knowledge as we seem to have is not enough; philosophy and logic as well as foresight and a great patience must be brought into the picture.

It is not enough, for instance, for our American colleagues to adduce proof both in America and Western Europe that the mortality from human tuberculosis declined simultaneously with the eradication of tuberculous cows, because it can be shown that human mortality declined as much elsewhere without the eradication of tuberculous cows, and even so declined while bovine tuberculosis, as in Queensland, was increasing.

Why Was the Question Raised?

No one could reasonably object to the propriety, and indeed the desirability, of drawing veterinary attention to the highly important fact that the great majority of veterinarians had overlooked, namely, the extraordinary differences that existed in the relationship of bovine to human tuberculosis in different parts of the world, especially between the Northern and Southern hemispheres.

The writer had found, wherever he went, that these differences either were ignored or were overlooked by the great majority of his fellow-veterinarians, so that the significance of these differences had not even been considered by the veterinary profession. A few medical men had observed and had commented upon these differences, but no veterinarian.

Thus, when in 1943 a Dr. Basil Sampson had a letter on the matter, as revealed in South Africa, published in the *British Medical Journal*, and a review of this letter in the *Veterinary Record* showed how little we knew about the question, sheer pique and pride in his own profession constrained the present

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writer to publish the results of his own research. Consequently, the writer feels that he has done no wrong in having tried to fill an immense gap in veterinary information. If, in his close study of a new and arresting set of facts he has erred in the matter of deduction, he can but appeal to his critics to give a better explanation.

If the logical deduction, for instance, from the fact that the decline in human mortality in Queensland has been greater than the decline anywhere else, notwithstanding that bovine tuberculosis abounds in Queensland, is not that exposure to the bovine germ is not necessarily an unfavourable factor, then what is the true deduction, and why has none of the writer's critics even stated the problem?

Why, in the face of this phenomenon of Queensland's relative immunity from human tuberculosis in the midst of bovine tuberculosis, have our colleagues in the United States concluded—as though they were living in another world—that the eradication of tuberculous cows and the pasteurisation of cows' milk was a universal prerequisite to the control of human tuberculosis?

Queensland is quoted as a convenient example, but the experience of Queensland is not unique; other areas in the Southern hemisphere show a similar human immunity in the presence of bovine disease.

Counsels of Perfection

It is because of such differences as those between North America and Queensland, and because there is no mathematically exact relationship between bovine and human tuberculosis, and because of practically ineradicable reservoirs of tuberculous infection among the social derelicts of almost all nations, and because of recurring social catastrophes such as wars, that the total elimination of tuberculosis must remain an unattainable dream.

In the United States, for instance, though every bovine beast were destroyed, there would still remain a vast negro population grievously infected with tuberculosis for which there are no reliable vital statistics. The increase in almost all countries of tuberculosis resulting from the total war cannot even be estimated, and there will be other wars.

Within a measurable time from now our reserves of fossilised hydrocarbons will be exhausted, necessitating great new distributions and destructions of populations. During such cataclysms the artificial control of such diseases as tuberculosis will be impossible and the ravages of those diseases against which we have been protected artificially will be terrible. And especially terrible, we may be scientifically sure, among those populations, human and animal, where susceptibility to specific infection has been exalted by such means as the artificial eradication of bovine tuberculosis.

Some New Evidence

Evidence of an exalted susceptibility to tuberculosis among cows, both in England and North America, may possibly be found in such recent reports as that of tuberculous infection of the udder among a number of cows in a tubercle free herd receiving injections for the treatment of mastitis. Or in the report from the United States of the tuberculous infection, and consequent destruction, of four herds of cows in succession from a single owner, suffering from tuberculosis. The latter report is alarming in its implication of both an exalted bovine susceptibility and the beginning of a real danger that the susceptible cow of the

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future may become a ready conveyor of human tuberculous infection. There is reason to think that such excessive phenomena are new, and that they may be a logical consequence of our massive interference with the natural mechanism whereby an ever-increasing resistance to tuberculosis has gradually been acquired by man and animal simultaneously and interdependently.

Formerly it was thought a disability and a misfortune that certain aboriginal peoples, for lack of prior contact with the blessings of civilisation, should be particularly susceptible to tuberculosis, but it would seem now that we are doing our utmost to exalt the susceptibility to tuberculosis not only of ourselves but of the cattle who have helpfully trekked with us along the evolutionary path towards a natural immunity.

A Preview of Two Worlds

Clearly, then, we stand, in the matter of the relationship of bovine to human tuberculosis, at a cross-roads. One road leads to a perfect mechanised disease-free world existing only in our imagination. In that world there shall be no more bacterial disease, of man or animal, so that the natural resistance to disease conferred by the painful but sure process of evolution will be superfluous.

Sure of the future, certain of freedom from war, convinced that the Machine age is here for ever, satisfied of the complete conquest by a single animal species of universal nature, we who propose to travel by this road towards Utopia think that we could now safely and logically discard all natural means, eradicate tuberculosis from all our cattle, swine, and birds, eradicate it, too, from the teeming millions of Asia and Africa, and so be sure that wherever we may be driven in the future, by those cataclysms of nature that even we cannot control, we shall in our state of exalted susceptibility never be exposed to infection. Total dreamland.

The other road, which is the road we shall have to travel, leads to an unknown future, but through dreadfully familiar country. Country ravaged by war, by soil erosion, by flood, country peopled by millions and ever more millions of human beings, submerged so deeply by inherited or environmental disabilities that disease beyond human hope of elimination must be their portion as far ahead as can be seen. Country not immediately or all at once bereft of underground and forest fuel resources, but ultimately so, whereby of grim necessity both man and animal will have to discover a fresh evolutionary path or perish from the earth. That is the real road, and it leads, so far as we can know, to the real world of the future.

The best provision we can make for such a world and against such a future would obviously be to hang on determinedly to such natural resistance as we have, and to such general means of fortifying that resistance as are naturally available. At a time when providence is out of favour it would, of course, not be expected that we should, for the sake of posterity, totally and unreasonably renounce all the artificial means we have elaborated for prolonging a twilight existence on this planet.

But at least we could postpone such biological practice as the eradication of bovine disease by means of the tuberculin test until it can be proved that the resulting exaltation of susceptibility is not loaded with dynamite. An abundance of vital statistics proves that so far as most of the Southern hemisphere is concerned such a postponement could safely be made.

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The danger of a fatal human tuberculous infection from bovine sources throughout the greater part of the Southern hemisphere is relatively small, and there is practically no risk of crippling disease, since bones and joints are not affected either in Australia or South Africa, so far as is known. In the Northern hemisphere the matter is different, and the writer does not need to be reminded of those everlasting two thousand deaths from tuberculosis of bovine origin annually in Britain, or of the many cripples from the same cause. Against these grim statistics, which strangely do not seem to be reduced by the ever-increasing pasteurisation of milk in Britain, there must be set, however, the fact that during the total war, in spite of the care for child nutrition in Britain, there has been a great increase in the incidence of tuberculosis of children from non-bovine sources.

Is this a consequence of exalted susceptibility, and will similar evidence for similar reasons be available in time in war-devastated Europe? And among negroes in America? If so, and if research into these statistics suggests that resistance to tuberculosis is growing less, then what could be more likely than that pasteurisation has something to do with it?

Nature Has the Last Word

As to the opinion of a writer recently on this subject that it is criminal to suggest that the human consumption of cows' milk containing tubercle bacilli may, on balance, do more good than harm, and that, if this were so, the proper course would be to add bovine germs to the milk, it should be clear that such advice is frivolous. In the present state of orthodox medical opinion one might as well expect prussic acid to be added to the children's milk, so why suggest it?

Moreover, we know nothing about the physico-chemical factors that might determine the effect for good or evil of such action, nothing of the relative virulence for such a purpose of infection direct from the animal or from an artificial culture of the organism.

All that has guided the writer in this study, and all that should guide us in any action we might take or refrain from taking, is what actually happens, as revealed by vital statistics, which are the recorded results of natural activity or unnatural interference with that activity.

Finally, the writer would summarise or define his thesis as, in effect, a plea for the abandonment of the test-and-slaughter policy for the control of tuberculosis or any other common disease of mammals on the grounds that it is in conflict with the nature of things, and consequently must fail in the end.

Vaccination reinforces and extends a natural process or tendency, and is thus in accordance with natural principles, but test-and-slaughter, which takes no account of individual excellence, violates natural principles. Carried to its logical conclusion and applied to the control of all bacterial disease it would wipe out every species, including man himself.

Thus test-and-slaughter, indiscriminately applied, is contrary to the categorical imperative of Kant, and is seen as a philosophic blunder of the first magnitude. At most it has only a limited and exceptional value: as a general and indiscriminate measure it stands condemned.

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THE DIAGNOSTIC VALUE OF ROTHERA'S TEST ON MILK

A. B. PATERSON,
Veterinary Laboratory, Ministry of Agriculture, Weybridge.

Introduction

ROTHERA'S test[1] is well known in human and veterinary medicine as a method of detecting acetone and acetoacetic acid in urine. It is performed by saturating the sample with solid ammonium sulphate, adding a few drops of dilute sodium nitroprusside solution, and then adding excess of concentrated ammonia. Development of a purple colour represents a positive test. Harding and Ruttan[2] found the test more sensitive for acetoacetic acid than for acetone, and that electrolytes other than ammonium sulphate could be used to increase sensitivity. The test is not given by β-hydroxybutyric acid, the compound which constitutes the major portion of the total "ketone bodies" in blood.

The value of the test on urine, as a diagnostic agent in cases of suspected ketosis of the dairy cow, is limited by its very sensitivity. Boddie,[3] Duncan and Huffmann,[4] and others, have observed positive reactions in normal stall-fed cows, and the test, as ordinarily carried out, may therefore be misleading. Partly because of this, and partly because a sample of milk is more easily obtained than a sample of urine, some practitioners apply the Rothera test to milk and take a positive reaction as confirming a clinical diagnosis of ketosis. The procedure has proved useful, but, so far as the writer is aware, little or no attempt has been made to examine the rationale of its use.

In the present inquiry the test was applied to milks containing added amounts of acetone, acetoacetic acid and β-hydroxybutyric acid in varying proportions, and factors which might affect the reaction were considered. Milk samples were examined from normal cows, from cows displaying clinical ketosis, and from cows suspected of this condition but in reality suffering from other disorders.

Terminology

The words "acetonæmia" and "ketonæmia" simply refer to abnormally high levels of acetone or of ketone bodies (acetone, acetoacetic acid and β-hydroxybutyric acid) in the blood, but since these bodies are merely an indication of disturbed metabolism and are not themselves the cause of clinical symptoms, the words should not be used as denoting a disease. The word "ketosis" refers to any condition marked by excessive formation of ketones in the body, but American veterinarians use the term to designate the bovine post-parturient hypoglycæmic ketosis described by Stinson[5] in this country, most commonly occurring about ten days after calving and associated with lack of appetite, wasting, fall in milk yield and other symptoms. The usage is loose, but the term "ketosis" is better than "acetonæmia," and as good as the older phrase, "post-parturient dyspepsia." There is, at present, no universally accepted term for the clinical condition, the etiology of which is still obscure.

Technique

In conducting the Rothera test in the present series of observations, 10 ml. of milk were pipetted on to 5 gm. of powdered ammonium sulphate in a test-tube and thoroughly shaken to ensure complete solution; 0.1 ml. of freshly-prepared 5 per cent. aqueous sodium nitroprusside was then added, followed by 2 ml. of concentrated ammonia, and the tube again shaken. The intensity of colour attained after five minutes was recorded in four arbitrary gradings, as (1)

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faint pink, feebly positive, or +; (2) definite pink ++; (3) purple, strongly positive, or +++; and (4) intense purple, or ++++.

It may be noted that 5 gm. of ammonium sulphate does not effect complete saturation, but is sufficient for the test and avoids the variable residue of crystals in the tube when indefinite excess is used.

A convenient modification for *field use* requires only the following simple equipment:—

(a) Test-tubes of $\frac{5}{8}$ in. internal diameter scratched with three marks, 1.7 in., 3.0 in., and 3.5 in. from the bottom.

(b) An intimate mixture of ninety-five parts of crystalline ammonium sulphate and five parts of coarsely ground sodium nitroprusside. If kept quite dry this mixture lasts indefinitely, but it is best to make up afresh every few months.

(c) A small bottle of strong ammonia.

Tubes may be carried ready filled to the lowest mark with the solid mixture. In making the test the suspected milk (preferably cooled under a tap to obviate subsequent frothing) is added to the second mark and the tube thoroughly shaken to dissolve the ammonium sulphate and nitroprusside. Strong ammonia is then added to the third mark, the tube again shaken, and the intensity of colour noted after five minutes.

In making the observations recorded below, determinations of "acetoacetic acid plus acetone" and of β-hydroxybutyric acid were made by the routine Weybridge procedure. This consists in precipitating the milk proteins with copper sulphate and lime, filtering, rapidly distilling (five minutes) an aliquot of filtrate (5 ml.) with sulphuric acid to obtain preformed acetone and acetone derived from acetoacetic acid, changing the tube receiver, adding dilute dichromate and continuing the distillation to obtain acetone derived by oxidation of hydroxybutiric acid. The acetone in each distillate is then estimated by photometric reading of the colour produced by alcoholic salicylaldehyde in strongly alkaline solution.

Rothera Reactions in Milk Containing Known Amounts of Ketone Bodies

The test was observed to be much less sensitive in milk than in aqueous solutions or in urine. β-hydroxybutyric acid added to milk in large amounts gave no reaction. The lower limit for detection of *acetone,* added to milk as standardised aqueous solution, was found to be about 5 mg. p.c. (milligrams per 100 ml. or parts per hundred thousand). At this level a faint pink, or + reaction, slowly appeared within five minutes. At a level of 10 mg. p.c. a definite pink, or ++ reaction, developed. A purple colour, or +++ reaction, developed at 15 mg. p.c., and an intense purple, or ++++ reaction, appeared at 20 mg. p.c. and above.

In determining the limits of sensitivity for *acetoacetic acid,* a dilute aqueous solution of the acid itself was prepared by shaking up equimolecular quantities of the ethyl ester and dilute caustic soda, allowing to stand for several days with occasional shaking, then saturating with sodium chloride and aerating until the aspirated air no longer gave an iodoform reaction with dilute alkaline iodine. Varying amounts of the solution (unstable) were added to normal milk, the Rothera test immediately performed, and the true concentration determined by the Weybridge method mentioned above. The lowest concentration of acetoacetic acid which could be detected by Rothera's test was about 1.5 mg. p.c., expressed as acetone. A definite ++ reaction was given at a little over 2 mg. p.c. At 3.5 mg. p.c. an intense purple, or ++++ reaction, was obtained immediately. The colours persisted for about an hour, after which they began to fade.

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It will be noted that in milk the reaction is approximately five times as sensitive for acetoacetic acid as for acetone.

The test was also performed on milks in which both acetone and acetoacetic acid were present, obtained by mixing samples from the preceding trials. As expected, the results showed that the intensity of the test depended not so much upon the total ketones present, as upon the relative proportions of acetoacetic acid and acetone. The lower limit at which positive reactions are obtained will obviously depend upon the extent to which the ratio between the different "ketone bodies" is constant or variable in milk from actual cases of ketosis.

Other Factors Affecting the Test

Milk samples containing added acetoacetic acid and acetone were allowed to stand at room temperature for two days, during which time they soured and clotted. Those containing acetoacetic acid showed diminished reactions on re-testing, presumably because of conversion of this compound to the less reactive acetone. Milks containing acetone alone were unaffected in respect of intensity of reaction.

In urine samples other electrolytes, such as sodium chloride, magnesium sulphate and sodium acetate, intensify the permanganate colour and may be substituted for ammonium sulphate,[6] but in milk samples this is not the case. Ammonium sulphate appears to be the only effective salt, presumably because in high concentration it completely precipitates interfering milk proteins.

Formalin in large amounts (10 per cent.) interferes with the test, but does not do so in any concentrations likely to be encountered in milk. Sulphonamides, linseed oil, strychnine, turpentine, iodine, camphor, or sodium salicylate do not give a reaction, and if used medicinally would not affect the test in milk.

Reactions Obtained in Untreated Milk Samples

(a) *Normal cows.*—Eleven samples of milk taken from a normal herd were analysed, on the day of collection, for β-hydroxybutyric acid and "acetoacetic acid plus acetone," the sum of these two fractions giving "total ketone bodies."

The average for total ketones was 2.5 mg. p.c., with a range of 1.4 mg. p.c. to 3.6 mg. p.c. The average for β-hydroxybutyric acid was 0.7 mg. p.c., the range being 0.4-1.7 mg. p.c. For acetoacetic acid plus acetone the average was 1.8 mg. p.c. and the range 1.0-2.8 mg. p.c. The β-hydroxybutyric acid constituted 18-37 per cent., average 30 per cent., of the total ketones, the remaining 70 per cent. being made up of acetoacetic acid and acetone.

This relationship is the reverse of that found in blood, in which β-hydroxybutyric acid makes up 70-80 per cent. of the total, and verifies an earlier observation of Knodt, Shaw and White.[7]

The Rothera test was negative in all cases, although the "acetoacetic acid plus acetone" was 2.8 mg. p.c. in one sample, 2.4 mg. p.c. in another, and 2.0 mg. p.c. in three more. In view of the fact that a positive reaction is given after addition of sufficient acetoacetic acid to normal milk to bring the analytical figure to 1.5 mg. p.c., the data suggests that the greater part of the fraction "acetoacetic acid plus acetone" is in the form of the less reactive acetone.

(b) *Cows suspected of ketosis but showing normal serum ketones on analysis.*—Milk samples from twelve such cows all gave negative results to Rothera's test. Analysis of the milk showed 0.9 mg. p.c.—3.6 mg p.c. total ketone bodies, a range similar to that given above for normal milk.

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(c) *Cows with post-parturient hypoglycæmic ketosis.*—Milk samples received by post from thirteen definite clinical cases and two doubtful cases all yielded positive reactions varying from + to ++++. Analyses showed that the two doubtful cases had values for total ketones below 5 mg. p.c., but that all the rest had values ranging from 13 mg. p.c. to 36 mg. p.c., with an average of 17 mg. p.c. The average value for "acetoacetic acid plus acetone" was 15 mg. p.c., with a range of 2.5 mg. p.c. to 30 mg. p.c. For β-hydroxybutyric acid the average value was 3.6 mg. p.c., and the range 1.8 mg. p.c. to 6.3 mg p.c. This last fraction corresponds to 10-40 per cent. of the total ketones, which, as in the case of the normal milks above, is much below the proportion found in corresponding blood samples (70-80 per cent.).

The fact that the ratio of β-hydroxybutyric acid to acetoacetic acid and acetone in milk is the reverse of that found in blood supports the finding of Shaw[8] that the mammary gland can utilise the former compound but neither of the latter compounds.

Comparison of the intensity of colour in the Rothera test with that to be expected from the analytically found amounts of "acetoacetic acid plus acetone," suggested that the greater part of this fraction consisted of acetone itself. For example, values such as 10 mg. p.c. only yielded a ++ reaction, whereas 2 mg. p.c. of acetoacetic acid alone, added to normal milk, produced a similar intensity. This suggestion was confirmed on two samples by determining total ketones before and after rapid aspiration with air to remove preformed acetone.

As mentioned above, however, these milk samples had been transmitted by post, and partial transformation of acetoacetic acid into acetone probably occurred in transit. It is likely that the proportion of acetoacetic acid would have been higher, and the Rothera reaction more intense, if the tests could have been carried out within a few hours of sampling.

Relationship of Ketone Bodies in Milk to Those in Blood Serum and in Urine

Various practitioners, notably Mr. Messervy at the time he was in Jersey, had at various times sent in simultaneous samples of milk and blood from cows suspected of ketosis to the Veterinary Laboratory at Weybridge. Study of the records of 268 cases in which total ketones had been estimated in both samples, showed that the level in the milk averaged about half that in the blood serum. Values in milk were of the order indicated above, figures of 15 mg. p.c. to 25 mg. p.c. being common. Five of the values even exceeded 50 mg p.c., the highest being 85 mg. p.c.

A series of cases of ketosis in which blood serum values were compared with urine values, showed that the latter frequently exceeded fifteen times the former, and that figures of the order of 1,000 mg. p.c. were quite common in urine. This concentration by the kidneys accounts for the frequency with which the urine of normal cows gives a positive Rothera test, and explains the observations of Duncan and Huffmann,[4] who found that out of 1,500 clinically normal cows examined by them 675 (45 per cent.) gave a positive urinary reaction.

Discussion

It is difficult to define the "normal value" of blood serum ketones in the case of the cow, since the same animal varies considerably throughout the year according to the method of husbandry in vogue. An average value for healthy cows at grass in summer is about 3 mg. p.c., or similar to the normal for man and

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other animals. For the same cows in stalls in winter the average value may be twice as high. Certainly any value up to 5 mg. p.c. may be treated as "normal," and values up to 10 mg. p.c. only represent a mild ketosis of little or no clinical significance. Indeed, ten out of fifty-nine healthy cows in a typical dairy herd examined by Eden and Green[9] showed serum ketones above 10 mg. p.c. under stall conditions in March, all of which fell below 5 mg. p.c. on turning out to pasture in May. One of these showed the very high figure of 45 mg. p.c. with no clinical symptoms beyond slight inappetence and milk yield a little below expectation. The milk was not tested, but on the basis of the findings now recorded a strong Rothera reaction would be expected. For the remaining nine animals a weakly positive reaction would probably have been shown. A positive Rothera test in milk is not, therefore, an infallible indication of a clinically recognisable ketosis. On the other hand, in all recorded cases accessible to the writer, now exceeding 300, in which clinical ketosis was suspected on grounds of dyspepsia and fall in milk yield, the level of milk ketones was such as to give a very definite Rothera test.

When used as an aid to diagnosis the position therefore appears to be :—

(a) Rothera test on milk negative even when positive on urine : ketosis definitely absent.

(b) Rothera test weakly positive : ketonæmia definitely present and the condition of ketosis confirmed if any clinical symptoms, such as fall in milk yield, are shown.

(c) Rothera test strongly positive; ketonæmia pronounced, although the severity of clinical symptoms may vary considerably and not be directly proportional to the intensity of the reaction.

Summary and Conclusions

(1) The Rothera test in milk samples is a valuable aid to diagnosis of that condition in cows described by Stinson as "bovine hypoglycæmic ketosis." Until more is known about post-parturient metabolic disorders the interpretation of a weak positive is ambiguous, because of the occurrence of mild ketonæmia in healthy stall-fed dairy cows, but a strong positive taken in conjunction with fall in milk yield is usually sufficient to define the condition.

(2) Because of the concentration of blood ketones by the kidneys the urine of a high proportion of normal cows gives a positive Rothera test, and the reaction on urine is therefore of little value as an aid to diagnosis. Milk samples are easy to obtain, the reaction itself is less sensitive in milk than in urine, and a definitely positive test is not shown until the level of blood ketones has risen to several times the normal value. The test on milk, therefore, deserves wider application than it has hitherto received.

(3) The level of total ketones in milk, as determined by chemical analysis, is usually about half that of the corresponding blood serum.

(4) Of the total ketones in blood, about 70 per cent. is usually β-hydroxybutyric acid and 30 per cent. "acetoacetic acid plus acetone." In milk the ratio is reversed, probably because the mammary gland can itself utilise β-hydroxybutyric acid.

(5) Of the fraction "acetoacetic acid plus acetone" in milk, a considerable proportion consists of acetone itself, and this proportion rises on long standing, by conversion of acetoacetic acid to acetone, so that the Rothera test becomes less intense as the milk sours. Samples should therefore be tested on the day

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they are taken. A convenient method of testing in the byre itself is described, using tubes already containing the required mixture of solid ammonium sulphate and sodium nitroprusside.

(6) Medicinal use of sulphonamides, strychnine, sodium salicylate, iodine, formalin, camphor, turpentine, and linseed oil, does not interfere with the Rothera test in milk.

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LAMENESS IN HORSES

By PROFESSOR J. J. O'CONNOR,
Donnybrook, Co. Dublin.

Definition

LAMENESS has been defined by Percival as the manifestation in the act of progression by one or more of the limbs of pain, weakness, or inability.

It may be added, however, that lameness can also be manifested at rest by an abnormal position of one or more of the limbs, e.g., pointing a fore-foot as in navicular disease, dropping of the elbow in radial paralysis, resting on the heels as in laminitis, frequent lifting of a limb as in arthritis.

Etiology

The cause of lameness in most cases is inflammation, acute, subacute, or chronic, asceptic or septic, but in several instances it is the result of paralysis from interference with the motor nerve supply of some of the muscles of locomotion. Occasionally it is mechanical in origin, following more or less complete anchylosis of joints, which, however, is a sequel to inflammation, being due either to the formation of new fibrous or osseous tissue at the level of an articulation or to the fusion of articular surfaces consequent on dry arthritis. In these cases of mechanical lameness pain may be present from a concurrent persisting inflammation in the affected region, a point which must be borne in mind when the question arises of cruelty for working a horse with an obvious mechanical impediment to the normal functioning of the limb, which is apt to be looked upon as the sole cause of the lameness. It may not be possible to demonstrate the presence of slight pain in such a case.

Lameness from an obvious cause is easily dealt with from the point of view of procedure, and it would appear that a treatise on the condition can be of little use, except it can afford considerable help in diagnosing the seat of the trouble in obscure cases. Some men are credited with the gift of lightning diagnosis, but the ground for such credit is not always reliable, being often based on the fact that improvement or cure of the lameness follows on the application of an antiphlogistic topic on the region diagnosed as affected, forgetting the *vis medicatrix naturæ*, irrespective of the part treated, rest and time being great healers of inflammatory affections.

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Experience in the examination of horses no doubt conduces to proficiency in the art of locating the part affected.

There are types of lameness, varying according to the region involved, which may be missed by the novice, and mistakes are made often enough by experienced practitioners not exercising sufficient care in the examination of lame horses. Hence there may be no need for an apology in referring to lameness in a methodical way from different aspects for the purpose of inducing veterinary surgeons, especially junior members of the profession, to devote more attention to the theoretical and practical study of lameness. A prerequisite to proficiency in the matter is a thorough knowledge of the anatomy and physiology of the limbs. It is obvious that an expert opinion cannot be given as to the presence of an abnormality if the clinician has not a clear idea of the normal structures and their functions. The student and budding practitioner should avail of every opportunity to observe horses in motion, including those working on roads and streets, and thereby become familiar with the gaits of horses, sound and otherwise.

There are various ways in which the subject of lameness may be considered to bring in some of the most important points concerning it, as follows :—

I. *Predisposing Causes,* which comprise :

(1) Nature of the work.

(2) Nature of the ground.

(3) Age of the animal.

(4) Heredity.

(1) *Nature of the Work.*—The hunter, on account of the speed at which he moves and the exertion of jumping, is subject to sprain and rupture of tendons, ligaments, and muscles, and to fractures. Draft-horses are liable to sprain of the flexor tendons and of the check ligament from the strain of heavy draft.

(2) *Nature of the ground.*—Fast work on hard ground favours concussion or jarring of the fore-limbs and the onset of sore shins and splints or general osteo perostitis in young horses. Smooth surfaces lead to slipping with over-distension of ligaments, tendons, and muscles, and to falls, with consequent injury to tissues, while stony, yielding or pebbly ground interferes with even tread and thus conduces to wrenching of joints and to stumbling.

(3) *Age of the animal.*—Young animals whose tissues are immature are obviously more prone to suffer from the effects of work than mature subjects. Hence the prevalence of sore shins, splints and phalangeal exotoses in the former.

(4) *Heredity.*—Physical weakness is undoubtedly hereditary, and in the case of the limbs must predispose to lameness, rendering the tissues less resistant to violent exertion and prolonged concussion, and, consequently, to such conditions as ring-bone, side-bone, spavin, curb, navicular disease and sandcrack, conditions which are generally accepted as hereditary. Faulty conformation and action of the limbs are transmitted from parent to offspring and lead to unequal distribution of weight on the limbs, and consequently favour lameness; for example, that caused by brushing in horses with turned-out toes, spavin and curb in badly shaped hocks.

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Temperament is another hereditary feature which may influence lameness, as in the case of a highly-strung or over-willing horse which exerts himself unnecessarily.

To the foregoing predisposing causes may be added unfit condition, that is, working a horse without due preparation, which leaves him more open to lesions of bones and tendons.

II. *Exciting causes* comprise :—

(1) *Traumatism,* including contusions and open wounds from falls, blows, kicks, brushing, speedy-cutting, ill-fitting shoes, pricks in shoeing, picked-up-nail.

(2) *Violent and repeated locomotory action,* affecting muscles, tendons, ligaments and bones and synovial sheaths.

(3) *Infection,* resulting, when local, in abscess formation or lymphangitis, adenitis or synovitis, and, when constitutional, like influenza, often causing sudden lameness, due to acute toxic synovitis of one or more synovial sheaths or joint capsules.

(4) *Rheumatism,* affecting muscles or joints.

The relative frequency of the various well-known forms of lameness is a matter of interest which can only be estimated approximately. Statistics published by the French army revealed that 60 per cent. of the total clinical cases were surgical lesions, and that of these lameness cases constituted 30 per cent. Lameness due to affections of tendons amounted to 33 per cent., of joints 30 per cent., of the feet 20 per cent., bones 15 per cent., muscles, nerves and vessels 4 per cent.

Lameness may be continuous, as in the case of that due to splint, ring-bone, or spavin, fugitive as in rheumatism, or intermittent as in the early stages of navicular disease and bobba bone. Lameness may diminish with exercise, as, for example, in navicular disease and spavin, but when the animal is rested for a while and again started the lameness is intensified.

Lameness may be hardly perceptible, distinct, or very marked.

Diagnosis of lameness includes recognising :

(1) The limb affected.

(2) The seat of the lameness.

(3) The nature of the lesion.

(1) *The limb affected.*—The horse should be examined at rest and during motion.

(a) *At rest* suspicion is aroused by an abnormal position of the limb. For example, in the fore-limb, pointing of the foot or flexion of the knee or fetlock, and, in the hind-limb, resting on the toe or heel or holding the limb abducted; also by shifting the weight from one fore-limb to the other, indicating that both limbs are affected, and by observing that the sound limbs are more under the centre of gravity than usual, to relieve the affected limb or limbs. It is well to look at the shoes to ascertain whether a shoe is excessively worn at a particular part, indicating unequal tread, e.g., the toe in spavin and navicular disease, and

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the heels in laminitis and low ring-bone. It is advisable to make the horse "come over" to see whether he favours one hind-limb in so doing, or shows evidence of shivering.

It is good practice if the horse has come a journey to leave him at complete rest for about half an hour before testing his action, because when the horse is then exercised lameness that has worn off during the journey will become apparent, such as that of navicular disease, spavin, and rheumatism.

(2) *During motion.*—Severe lameness is observed at the walk and even slight lameness at the trot, but at the gallop the pace is so rapid that the lameness is masked.

Fore-limb lameness at the trot.—To test for this the horse should be led with a halter or snaffle bridle, with a free head, on hard, level ground, without clothing or harness. It is easier to detect movements of the head in unison with the incidence of weight on the limbs when the horse is trotted in hand than when mounted, but when there is doubt as to the presence of lameness it is well to have the horse mounted to increase the weight on the limbs and perhaps make slight lameness more manifest. When mounted the movements of the head are somewhat interfered with by the action of the reins and the movements of the rider. When the horse is being led the reins should be held about 18 inches from the bit and the man should run on without looking back at the horse, and if he must use his stick he should do so with his left hand behind his back. The horse should never have sufficient rein to enable him to turn this heels on the leader.

The examination should be carried out in a quiet place where there is nothing to distract the horse and make him forget about the lameness. He should be carefully watched when first trotted after resting lest ephemeral lameness may disappear with exercise. Particular notice should be taken of the animal during turning when extra weight is placed on one of the limbs, causing more marked limping when it is the lame limb that is bearing the weight.

When the horse is trotted on pavement or concrete the noise of the feet serves as a guide by noticing the difference in sound made by the lame and sound limbs. Shoulder lameness is more readily detected on soft, yielding ground, as it interferes with advancement of the affected limb. It is hardly necessary to say that the head drops or nods when the sound limb bears weight, and rises, with some deviation towards the opposite side, when the affected limb makes contact with the ground, the reasons for such movements being, obviously, to minimise the pain suffered by the animal. The movements of the head and the way in which weight is placed on the limbs should be observed at the same time. Keeping the ears in view is useful when the horse is being trotted from the observer to see whether their level changes from up and down movement of the head. In trivial lameness it may be impossible to recognise head oscillations. The examiner must not be misled by "bridle lameness," that is the animal appearing to be lame from the side from which he is led. When in doubt about this he should have the horse led from the opposite side. The wide-breasted cart-horse rolls in his gait and may appear to the novice not to be going level.

Hind-limb lameness.—In this the oscillations of the head are not so extensive. In many cases no nodding is noticed. The head is generally lowered with sup-

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port on the opposite fore-limb. The sound quarter sinks and the head rises when the lame limb comes to the ground.

Seat of the lameness.—It sometimes requires patient and methodical searching to discover the seat of the lameness, and in some instances it is not possible to locate it, even after a careful process of elimination.

The first stage in the procedure is to get a complete history of the case and to make inquiries, the answers to which may throw light on the subject. It may be said at the outset that the foot should be the first region to be examined even when there is an obvious lesion in another part of the limb, and it must be remembered that inflammation inside the hoof may be the origin of heat and swelling in the metacarpal or metatarsal region, giving the impression that the latter is the seat of the trouble.

Mistakes have been made by veterinary surgeons ascribing lameness to some abnormality, such as splint, spavin, or ring-bone, when it is really due to pus in the foot or a picked up foreign body there which has been overlooked.

The foot having been eliminated, a methodical examination of the limb should be made of its anterior, lateral and posterior aspects from the shoulder, or hip, to the hoof, bearing in mind the recognised conditions which are common sources of lameness.

This examination comprises:

(1) Inspection.
(2) Palpation.
(3) Passive movement of joints.
(4) Observation of posture.
(5) Observation of gait.

(1) *Inspection.*—The limb is viewed from various aspects, comparing it with its fellows to ascertain whether any deviation from the normal catches the eye as regards conformation or pathological conditions.

(2) *Palpation* should be carried out methodically on the front, sides and back of the limb, searching for anatomical abnormalities and evidence of inflammation in the form of heat, pain, and swelling, and applying pressure in the suspected region to ascertain if the horse will flinch. Regarding evidence of pain on compression, the same amount of compression should be applied to a corresponding part on the sound limb, as a control, for a sensitive animal may yield to this test on a normal part.

Special attention should be given to the seats of splint, ring-bone, bobba bone, spavin, curb, tendinitis and synovitis, brushing and speedy-cutting, and in the hind-limb rectal examination is often indicated to eliminate fractured pelvis, iliac thrombosis, etc., and to avoid the mistake sometimes made of treating a horse for spavin when affected with fractured pelvis.

(3) *Passive movement of joints.*—Passive flexion, extension, abduction, adduction and rotation of joints as a test for evidence of inflammation in articular and peri-articular structures is a useful aid in diagnosis, but the same movements must be practised on the sound limb for the purpose of comparison. Passive flexion of an inflamed joint always intensifies the lameness.

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(4) *Observation of the posture* of the animal at rest has been alluded to as varying according to regions affected, e.g., that assumed in radial, crural, and extensor popliteal paralysis, contracted tendons, laminitis, etc.

(5) *Observation of the gait.*—The gait of the horse may point more or less to the cause of the lameness, e.g., difficulty in taking the limb forward in shoulder and hip lameness, holding a joint stiffly when its region is involved, improving with exercise as in navicular disease and spavin and rheumatic affections, going on the heels in laminitis, the peculiar gaits of supra scapular paralysis and rupture of the flexor metatarsi muscle.

Too much reliance must not be placed on difficulty in advancing the limb as indicating an affection of its upper region, as the same gait may be observed in lesions of its lower part. The veterinarian must not be misled by the owner confidently saying that the horse is lame in or has hurt the shoulder, as this is a common diagnosis of the layman even, in some instances, when there is found to be a nail in the horse's foot. Incidentally, it is well to remark that even after an apparently careful examination of the foot with knife, hammer and pincers the veterinarian may be on the point of concluding that it is not affected, when persevering further reveals pus beneath the horn. It is important not to come to a hasty conclusion in agreement with a diagnosis already made by a recognised authority, for it could possibly be wrong.

Nerve blocking is indicated when there is no objective symptom revealing the source of the lameness, the plantar nerves being blocked just above the fetlock to eliminate the foot, and the median and ulnar nerves to eliminate the region between the seat of its injection and that of the plantar nerves.

When the foot has been desensitised it is dangerous to exercise the animal at a fast pace on hard ground, because by so doing one of the phalangeal bones or the navicular bone may be fractured.

(3) *The nature of the lesion.*—Once the seat of the lameness has been determined it is generally easy to recognise the nature of the lesion, to estimate its gravity and to deal with it accordingly.

Abstracts from Current Literature

PENICILLIN IN ANTHRAX*

THE success attained in the treatment of human cases of cutaneous anthrax with penicillin at the Pennsylvania Hospital for Contagious Diseases is of interest in veterinary medicine. Although the characteristic and almost universal bacteremia of anthrax in animals was absent, the rapid disappearance of the specific agent (*Bacillus anthracis*) from the local lesion established its sensitivity to the microbicide action of the drug. The study also revealed the strange contrast that in vitro *B. anthracis* was 100 times more resistant to penicillin than *Staphylococcus aureus*. In three cases favourable response to penicillin was evoked from the injection of 100,000 to 475,000 units. At least a total of 200,000 to 400,000 units of penicillin, given at the rate of 100,000 units in 24 hours, should be administered to the average adult to obtain prompt and satisfactory therapeutic response.

* *Journal of the American Medical Association,* December 9, 1945.

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METAZOAN PARASITES IN MEAT INSPECTION*

A PLEA is made in Canada for higher standards of efficiency in meat inspection and sufficient trained personnel to bring all slaughterhouses under its directives. Only 60 per cent. of Canadian abattoirs are operated under the Dominion meat-inspection service; the other 40 per cent. are free to market their output without adequate sanitary supervision. Particular consideration should be given to meats and meat products sold to be eaten without further cooking, i.e., cooking of such food should be properly supervised in the processing plant. The parasites encountered in the routine meat inspection of Canada, which render parts or all of infected carcasses unfit for food, are:—

(1) The cystic form of the human tapeworms, *Tænia saginata* and *T. solium*, which cause cysticercosis (measly beef or pork). The meat inspector finds these in the masseter, pterygoid or/and intercostal muscles, or in the heart or diaphragm.

(2) The cystic form of the canine tapeworm, *Echinococcus granulosus*, which causes large hydatid cysts in the abdominal organs of man and animals. Dogs having access to the viscera of slaughtered animals at small abattoirs are a common means of spreading the infection.

(3) From 40 to 50 per cent. of Canadian sheep are infected with the tapeworms *Monezia expansa* and *Thysanosoma actinioides*. Though not transmitted to man, they render livers offensive for human use.

(4) *Trichinella spiralis* infection, though not detectable in the routine of meat inspection, imposes the responsibility of insuring proper cooking of processed food which would be eaten without further cooking.

The other more common metazoa detrimental to animal production, and which, therefore, reduce food poundage, are: *Ascaris suis, Dictyocaulus filaria*, and *Metastrongylus apri* of swine (the causes of verminous bronchitis); *Œsophagostomum columbiana*, which causes nodular disease of the intestine; *Cœnurus cerebralis*, or larvæ of the canine tapeworm, which causes gid in sheep; the larvæ of *Œstrus ovis*, or grub of the facial sinuses and lower air passages in sheep; and ox warble, or larvæ of the heelfly (*Hypoderma lineata*).

* *Canadian Journal of Comparative Medicine and Veterinary Science*, January, 1945.

DIFFERENTIATION OF BACILLUS ANTHRACIS FROM NON-PATHOGENIC AEROBIC SPORE-FORMING BACILLI†

STEIN, of the U.S. Bureau of Animal Industry, studied comparatively the cultural, biochemical and pathogenic properties of 53 strains of *Bacillus anthracis* and 64 strains of similar (anthrax-like) sport-forming organisms, such as *Bacillus cereus, siamensis, subtilis, tropicus, mycoides*, etc., which in themselves are not reliably distinguishable from *B. anthracis*, independent of their pathogenicity. Old cultures of *B. anthracis* (11 years) in doses of 0.25 c.c. were virulent for guinea-pigs, whereas doses eight times larger of the other strains were non-pathogenic for this laboratory rodent. However, tests for motility, reduction of methylene blue, hæmolysis, fermentation of salicin, rapidity of gelatin liquifaction, action on litmus milk and colony appearance, furnish useful evidence in making differentiations between *B. anthracis* and the common, ærobic spore-forming bacilli.

† *American Journal of Veterinary Research*, January, 1944.

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Irex (Champion).

Foals must be broken to halter early in life.

EFFECTS OF ARTIFICIAL LIGHT ON GROWING CHICKS*

CHICKS hatched and brooded during the hot summer months in California were checked for gains and feed consumed under (1) artificial light all night, (2) artificial light from midnight to daylight, and (3) no artificial light. At the end of ten weeks in one experiment and twelve weeks in five other experiments, the chicks receiving artificial light weighed more and had eaten less than those raised without any artificial light. In four of the experiments the cockerels were separated from the pullets at the age of twelve weeks. Under artificial light from midnight to daylight or all night they weighed more at the ages of sixteen, twenty and twenty-four weeks than those not receiving artificial light. The weight of the ones exposed to artificial light all night was slightly less than that of the midnight-to-daylight group.

Artificial light, however, had little effect on the average time the pullets began to lay or upon the weight of the first ten eggs they laid.

* *Poultry Science*, November, 1944.

Reviews

The Authentic Arabian Horse and His Descendants. By Lady Wentworth. George Allen and Unwin, Ltd. Price £5 5s.

LADY WENTWORTH has again presented us with a masterpiece, a worthy successor to her previous work, "Thoroughbred Racing Stock." The Arabian horse has been the theme of poem and story for countless generations. This beautiful animal has been wrapped in an aurora of sentiment and romance, and even reverence, that places it on a par with legendary figures. Yet in spite of all this, this superb beast can claim distinction above all others of his species and support his claim on fact. His story is one of triumph against adverse circumstances. He has existed and prospered in the most inhospitable lands in this world, he has remained for centuries true to type, and by conquering nature's adversity developed a character, a stamina and endurance second to none, and, moreover, he can transmit such qualities. He can truly be called the King of all Horses, for is he not the source of all the true blue blood of the thoroughbred equine world?

Lady Wentworth has an unrivalled knowledge of all these matters, and she has done a great service by placing her factual knowledge on record. No one else could have done this as well, for is she not the direct descendant of that remarkable couple, Lady Anne and Wilfred Scawen Blunt? The Arabian world, both of man and beast, owes much to them and to the author.

The book is divided into four sections, the first dealing with historical essential facts and fictions and the life of Lady Anne and her husband. In the second we are taken to Arabia and adjacent lands, and in our reading we breathe the very soul of the desert, its life, its sport and vicissitudes. In the third section we see the influence of the Arab on the outside world, and also in this part there is much of interest on riding, breaking and care of horses.

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In the last section one reads with some emotion the last words of Lady Anne, and there are many statistical facts placed on record.

It is extremely difficult to do full justice to such a book in a review, for it teems with facts and interest from cover to cover.

It is a book that one will turn to again and again, and one which, we are sure, will find an honoured and much frequented place in the library of all who are interested in the Arabian horse, Arabia and the Arabians.

It is no book to be read and cast aside, but rather one to which one will turn again and again. The veritable Bible of the Arabian Horse.

The author and her publishers are to be congratulated on the beautiful format; it is many years since one had the pleasure of handling such exquisite craftsmanship. The illustrations alone, 24 of which are in colour, are in themselves sufficient to give many hours of pleasure to all those who love a horse.

Rabbits' Ailments: A Short Treatise on the Domestic Rabbit in Health and Disease. By MAJOR W. P. BLOUNT, Ph.D.(Edin.), F.R.C.S. Published by *Fur and Feather*, Idle, Bradford; 1945. Price 3s. 6d., post free.

THIS is a most useful and timely publication. The emergency of war has led to a great increase in the domestic rabbit population, and it is more essential than ever that breeders and rearers should have, at any rate, a reasonable elementary knowledge of the anatomy and physiology of their charges, and be able to recognise deviations from the normal, in order that they may keep their rabbits at a high economic level. Those who have experience in this field know that there is a great wastage from disease, and also on account of defects in the feeding and general care and hygiene of rabbits. Any efforts to educate the owners in what can be called rabbit husbandry in its widest sense meets with our commendation, for thereby the efficiency of the industry can be greatly improved.

This was evidently the object in the mind of the author. The book is arranged in four parts. Part I, " The Rabbit in Health," gives a lucid account of the essential features of the structure and function of the body from birth to maturity. The part dealing with coprophagy is particularly interesting, in view of the discussions which have taken place recently anent this phenomenon.

Part II introduces the reader to the abnormal, i.e., disease, common ailments, how they may be recognised, and the value and methods of making post-mortem examinations.

Parts III and IV deal with the specific and non-specific diseases.

It may be that these last two sections are a little above the head of the ordinary rabbit-keeper, but the methods of approach and descriptions are such that they should be intelligible to a person versed in the elements of biological science. The book will also appeal to the professional man, graduate or undergraduate. They will read it with interest and profit, for coincident with the increasing importance of the domestic rabbit, the rabbit-keeper turns more and more to the professional man for advice and assistance.

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